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Auditory Brainstem Potentials: Comments on Their Use During Infant Development*

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The review article by Salamy (1984) provides a perspective on the significance of auditory brainstem potentials in the newborn and developing infant with particular emphasis on their use in quantifying and predicting development disorders. Dr. Salamy's methods of analysis involve measuring the latencies and amplitudes of the components of the auditory brainstem potentials during maturation and noting the effects of changes in certain stimulus parameters, such as rate. With these measures, Dr. Salamy believes that it is possible to distinguish and differentiate the group of children at risk for hearing and neurological disorders from those who will develop normally. If this premise were true, it would be a major contribution to the care of the newborn using the relatively simple and noninvasive technique of auditory evoked potentials.

However, I am not as optimistic as Dr. Salamy that the proposed measures of auditory brainstem potentials can provide the information for accurate prediction of the subsequent outcome of infants because of several limitations that Dr. Salamy himself considers.

First, the methods of recording have limitations. Dr. Salamy has selected from the auditory brainstem potentials three principal waves from which he makes measurements, i.e., waves I, III, and V. These components are elicited in a paradigm in which binaural stimulation is used and in which the recording is between the vertex and one of the mastoids or earlobes. Binaural stimulation assumes that the functioning of each cochlea is intact and that the central auditory pathways are symmetrically active. Children with altered unilateral central brainstem or cochlear function might be underestimated because stimulation of the unaffected ear using binaural signals would elicit normal components. Furthermore, since both recording sites are "active," their differential recording produces a mixture that may not reflect lateralized differences in the brainstem. Interpretation of auditory brainstem potentials would be simpler if the recordings were between the vertex and a noncephalic reference. I would prefer that monaural stimulation also be employed as a screen of unilateral cochlear and brainstem disorders.

*This review is complementary to that of A. Salamy, *J Clin Neurophysiol* 1984;1:293–329.

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Second, there are important stimulus factors that can influence the definition of the brainstem potentials. The polarity of the stimulus can affect the shape and relative amplitudes of the components particularly to high intensities. Stimulus rate also has an important differential influence on the components. Furthermore, stimulus intensity has significant effects on the identification of the components. Specifying a click in terms of "normal hearing level" is not sufficiently rigorous. I would recommend that the stimulus be defined by its physical parameters, i.e., "dB peak equivalent SPL." Finally, recording parameters such as filter widths and slopes will alter the morphology, amplitude, and latency of the components. Thus, any test that has clinical significance must rigorously define the effects that these stimulus and recording factors have on the brainstem components. Dr. Salamy has not yet studied in sufficient detail the effects that many of these variables have on measures of auditory brainstem potentials during development. Extensive generalities from Salamy's results will only be applicable when the effects of these variables can be ascertained.

Third, the major definition of abnormality in auditory brainstem potentials has depended on latency measures. Stockard (1983) has shown that the absolute latencies and central conduction times do follow a normal or gaussian distribution. Thus, the definition of abnormality in a one-tailed test can be between 2 and 3 SDs from the mean for a 97.5 to 99% confidence limit. However, for amplitude or amplitude ratios, the distribution of measures does not follow the gaussian rule. This is evidence even in Salamy's own observations in which wave V could be nonexistent in normals if 2 SDs from the mean were utilized. Nonparametric statistics must be employed for these kinds of measures to define confidence limits. These statistical issues become particularly important in attempting to use auditory brainstem potentials to define an abnormal infant. Salamy's results suggest that the *group* of normals and the *group* of abnormals can be statistically distinguished using certain measures of these potentials. However, what is not stated is that it would be very difficult to use the amplitude measures or the trajectories of latencies and amplitudes to define abnormality in an *individual* infant. The overlap between the normal and the at-risk population is sufficiently large to make the test statistically unreliable for the individual subject. What is needed is a measure of the auditory brainstem potentials that can predict "abnormality" for an individual infant.

Fourth, knowledge of what structures generate the components will affect the use of the test. The present concept that a lesion of a particular site in the brainstem causes a particular alteration in specific components of the potentials and thus is the site of the generators is too simplistic. For instance, in animal experiments it is clear that both a lesion of the trapezoid body and a lesion of the superior olive contralateral to the ear being stimulated, produce identical changes in wave III of the evoked potential (Wada and Starr, 1983). This does not mean that both the trapezoid body and the superior olive are the generators for wave III but, rather, that the trapezoid body conveys impulses to the superior olive which, in turn, may generate wave III. The definition of an alteration in wave III does not distinguish where the lesion exists along the auditory pathway, i.e., trapezoid body or the superior olive. Furthermore, it is apparent from the recent work of Sohmer et al. (1983) that the auditory brainstem potential generators are an extremely hardy group of elements. They resist hypotension and anoxia to an extent that is remarkable. The neural generators are obviously immune to the usual factors

that would affect synaptic function such as anoxia and anesthesia. This does not mean, necessarily, that nerve fibers are the structure generating auditory brainstem potentials. Rather, auditory brainstem potential generators are metabolically very different from other neural elements within the nervous system and an awareness of the type of generators may affect the generalizations that will be made from this test.

Fifth, the considerable importance of cochlear and peripheral factors for auditory brainstem potentials is not emphasized. The role of peripheral factors in contributing to the various components of the auditory brainstem potentials will continue to expand. For instance, Don and Eggermont (1978) have clearly shown that each portion of the basilar membrane contributes to wave V, whereas only the more basal end of the cochlea contributes to wave I. A differential development of the cochlear structures contributing to waves I and V might be an essential feature affecting auditory brainstem potential development during maturation rather than changes in the central pathway as Salamy suggests.

Sixth, I am not sure that the newly developed three-dimensional methods for depicting auditory brainstem potentials (Pratt, 1984) will enhance our understanding of the generation of these components or even their clinical utility as Salamy predicts. While there may be certain instances in which these new approaches will have application, the current practice of measuring latencies and amplitude ratios of individual components will probably continue to be utilized in the clinic. More refined methods to enhance quantification such as soft-wave programs to define signal-to-noise ratios and the reliability of evoked potential recordings will be of practical use in the clinic. One must always bear in mind that the components comprising the auditory brainstem potentials are of very low amplitude and masked by the background EEG "noise." The events, to be averaged, must be securely time-locked to the stimulus and, if variability were to occur, the components could not be defined. It may be that the loss or alteration of an evoked potential component reflects the development of temporal jitter in its generation rather than a loss of the component itself. One must always remember that averaging has significant limitations for studying time-varying neural events.

In summary, Salamy's article presents a perspective on the application of auditory brainstem potentials in the developing infant. He presents some important preliminary observations. Their value will be enhanced as (1) methods of stimulus and recording are standardized, (2) apppropriate statistical methods are developed to define "abnormality," (3) knowledge of the neural generators of the components expands, and (4) understanding of the effects of maturation of the cochlea and the central auditory pathway increases.

Auditory brainstem potential measures have developed considerably since the pioneering work of Jewett (1970) and Sohmer and Feinmesser (1967). We must remember, however, that this method, while of benefit, has significant limitations. It measures principally sensory capacities of the auditory system that may have only a limited influence on the eventual cognitive and behavioral outcome of the infant. Attempts should be made to relate evoked potential measures to other methods correlating brain structure and function such as the nuclear magnetic resonance, positron emission tomography, and blood flow. In the development of normal function the brainstem plays an important but probably not an indispensable role. Dr. Salamy is to be commended for his enthusiasm in trying to use a measure of brainstem function,

auditory brainstem potentials, to isolate those infants who have a high risk for being abnormal.

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